Southern C Particle Center and Supersite
UCLA ✧ USC ✧ Rancho Los Amigos ✧ UC Irvine ✧ UC Riverside ✧ UC Davis ✧ Michigan State
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October 14, 2003

Dave D. Lauriski
Assistant Secretary of Labor for
Mine Safety and Health
Room 2313
1100 Wilson Boulevard
Arlington, VA 22209-3939
Fax #: 202-693-9441

Dear Assistant Secretary Lauriski:

I write in response to the notice of proposed rulemaking published by the Mine Safety and Health Administration's (MSHA) on August 14, 2003 to revise the health standard to protect underground metal and nonmetal from diesel particulate matter (DPM). *(Federal Register, Vol. 68, No. 157, 48668-48721).* Under California law (AB1807) chemical compounds are identified as "Toxic Air Contaminants" when they "may cause or contribute to an increase in mortality or in serious illness, or which may pose a present or potential hazard to human health". In the summer of 1998 the California Air Resources Board (CARB) identified DPM as a Toxic Air Contaminant following a lengthy review process that lasted for approximately 9 years. AB1807 requires the literature reviews and determinations of the State Agencies proposing listing of a chemical substance be reviewed by an expert scientific committee, the Scientific Review Panel (SRP), to determine the scientific adequacy of the proposed listing. The SRP is comprised of outstanding scientists from California Universities and I chair the Panel.

By way of introduction, in addition to chairing the SRP, I am Professor of Toxicology in the UCLA School of Public Health. I direct the Southern California Particle Center and Supersite (SCPCS), one of the five Centers funded by U.S. EPA to address health effects of airborne particulate matter. I also Direct the UCLA Center for Occupational and Environmental Health, the Asthma Consortium, and the UCLA Environmental Tracking Program. I have served as Deputy Director of the National Institute for Occupational Safety and Health (NIOSH), Director of Toxic Substance Standards at OSHA, and I currently Chair the Subcommittee for Carcinogens of the Board of Scientific Counselors of the National Toxicology Program although my term ends this week. I have attached my NIH biosketch for your consideration.

The findings of the SRP from its deliberations of DPM are attached to this letter. The SRP concluded "Based on the available scientific evidence, as well as the results of the risk assessment, we conclude that diesel exhaust be identified as a Toxic Air Contaminant (TAC)". The ! : from structure/activity

AB29-comm-24

considerations, the genotoxicity of DPM, animal studies and the extensive epidemiological evidence demonstrating increased risk of lung cancer. In addition the SRP noted there is extensive evidence for non-cancer effects of DPM including adjuvant effects of DPM in relation to asthma. During its deliberations the SRP held a special meeting to review the evidence for the carcinogenicity of DPM and invited the leading scientific experts in the field to address the Panel. When asked directly whether DPM should be listed as a TAC, every invited scientist agreed. There has been widespread debate on the toxicity and carcinogenicity of DPM; the California process was slow and very deliberate. In the end, it was apparent the evidence was overwhelming especially the human epidemiological evidence from a range of occupational studies including truckers and railroad workers.

An area of controversy surrounding the identification of DPM as a TAC in California related to the risk assessment conducted by the State Agencies. There has been considerable discussion about the risk assessment, and the debate reflects the limitations of the methods available as well as the significant impact that rulemaking may have on affected industry. The SRP concluded that a risk of $3 \times 10^{-4}/ug/m^3$ is a reasonable estimate of the unit risk value. This value was derived from two separate approaches which yield similar results and this number is consistent with the value estimated by an advisory committee to U.S. EPA in 1979 based on a comparative risk assessment approach. Thus, the risk estimates for DPM have been relatively consistent for a considerable period of time.

Subsequent to the deliberations of the SRP on DPM a number of additional studies have added weight to the conclusions of the Panel including papers by Stayner et al (American Journal of Industrial Medicine, 34:207-219 (1998) and Steenland et al (American Journal of Industrial Medicine, 34:220-228 (1998). In addition, there have been dozens of new papers on the toxicology of DPM in relation to cancer and non-cancer effects. I will provide a list of references under separate cover. The conclusion is clear: the evidence for the carcinogenicity and non-cancer health effects of DPM has grown since 1998 and reinforces the decisions made by the State of California. The scientific evidence for the health effects of DPM is overwhelming, and while there may be issues of control that I cannot address it would be a serious error to not consider the evidence on the toxicity of DPM to be more than sufficient for regulatory purposes. I can think of no other substance where the evidentiary database is more compelling.

Research being conducted within the SCPCS mentioned earlier reinforces the conclusions about the serious adverse health effects associated with the non-cancer endpoints. We have demonstrated mitochondrial uptake of ultrafine particles from roadways and subsequent destruction of the impacted mitochondria. We have demonstrated strong redox activity and reactive oxygen species production from DPM with the subsequent potential for carcinogenesis and affects on allergic airway disease. We have demonstrated adjuvant effects of DPM on asthma. We have evidence for developmental effects, low birth weight and preterm birth in relation to traffic density. We are aware of the mutational spectra associated with DPM exposure in the "big blue rat" which further demonstrates the genetic toxicity of DPM. In short, the evidence continues to grow that

DPM is associated with adverse health effects; there has been no diminution of evidence of the consequences of DPM exposure.

I have had extended discussion with engine manufacturers about the health effects of diesel and the ability to produce engines which effectively control particulate matter and vapor phase co-pollutants. Most recently at a CARB sponsored meeting (Haagen-Schmidt) I discussed the issues with representatives of European diesel companies. It was my impression based on the informal conversations that there is significant progress being made to develop pollution free diesel engines. The representatives were very outspoken and positive about recent developments. In my view MSHA should continue its regulatory initiative with respect to DPM, since the result will be continued development of innovative technology and the ability to reduce worker exposure levels even further. Miners must be protected from the well-known health risks from exposure to DPM. The health effects associated with DPM exposure are real, and the focus of attention should not be on that threshold issue, but rather on the potential for technological advances. The emphasis at this stage should be on the control of DPM not reverting to time-worn debates about the science, and the greatest attention should be on the new, developing technology that is becoming available.

I appreciate the opportunity to comment on the issue of DPM. I am available for further discussion if greater detail would be helpful.

Sincerely,

John R. Froines, Ph.D.
Professor and Director

UNIVERSITY OF CALIFORNIA, LOS ANGELES

BERKELEY • DAVIS • IRVINE • LOS ANGELES • RIVERSIDE • SAN DIEGO • SAN FRANCISCO

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SANTA BARBARA • SANTA CRUZ

DEPARTMENT OF ENVIRONMENTAL HEALTH SCIENCES
SCHOOL OF PUBLIC HEALTH
BOX 951772
LOS ANGELES, CALIFORNIA 90095-1772

May 27, 1998

Mr. John D. Dunlap, III
Chairman
Air Resources Board
2020 L Street
Sacramento, California 95814

Dear Chairman Dunlap:

I am pleased to forward to you the Scientific Review Panel (SRP/Panel) Findings (enclosure) for the Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant Report as adopted unanimously at the Panels April 22, 1998 meeting.

The data, developed and reviewed by OEHHA and ARB, in the scientific risk assessment on exposure to diesel exhaust (Part A) and its health effects (Part B), are extensive and scientifically sound. The SRP notes the report documents the fact that diesel exhaust includes over 40 substances listed by the U.S. Environmental Protection Agency as hazardous air pollutants and by the ARB as toxic air contaminants.

The exposure estimate in the report may underestimate many Californians actual total exposure because it excludes elevated exposures near roadways, railroad tracks, and inside vehicles. Other routes of exposure to diesel exhaust, such as ingestion and dermal absorption are also excluded.

Development of this report began in 1989, and this compound has the most human epidemiological studies (over 30) than any of the previous 21 toxic air contaminant reports the Panel has reviewed. These studies have investigated the relationship between occupational diesel exhaust exposure and lung cancer, and the epidemiological evidence indicates exposure to diesel exhaust increases the risk of lung cancer. It is noted that in 1990 the State of California, pursuant to Proposition 65, identified diesel exhaust as a chemical known to the State to cause cancer.

There are a number of adverse long-term noncancer effects associated with exposure to diesel exhaust. These effects include chronic bronchitis, inflammation of lung tissue, thickening of the alveolar walls, immunological allergic reactions, and airway constriction. As new quantitative data emerge from research on adverse noncancer effects from diesel exhaust, the Reference Exposure Level may require adjustment.

The Panel believes there is still more to be learned about the adverse health effects associated with exposure to diesel exhaust. The Panel is concerned that some technological advances may result in greater total particulate exposure, particularly of fine particles that penetrate deeper into the lungs, but some controls and fuels may reduce overall particulate level. The Panel encourages further research to quantify the amounts of specific compounds emitted from a variety of engine technologies, operating cycles, and fuel to characterize better any differences between old and new fuels and technologies.

The Panel recognizes that diesel exhaust is a mixture of compounds and the potency factor may change as a result of new engine technologies and cleaner fuel. Accordingly, the unit risk factor may change as a result of new peer reviewed research.

We welcome any opportunity to provide additional information helpful to you or that would facilitate the process of identification.

We would appreciate our Findings and this transmittal letter being made a part of the final report.

Sincerely,

John R. Froines

John R. Froines, Ph.D.
Acting Chairman
Scientific Review Panel

Enclosure

cc: Scientific Review Panel Members
Michael Kenny, ARB
Bill Lockett, ARB

Findings of the Scientific Review Panel on
THE REPORT ON DIESEL EXHAUST
as adopted at the Panel's April 22, 1998, Meeting

Pursuant to Health and Safety Code section 39661, the Scientific Review Panel (SRP/Panel) has reviewed the report *Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant* by the staffs of the California Air Resources Board (ARB or Board) and the Office of Environmental Health Hazard Assessment (OEHHA) describing the public exposure to, and health effects of, diesel exhaust. The Panel members also reviewed the public comments received on this report.

Panel members participated in workshops devoted to discussion of the exposure and health issues associated with diesel exhaust in September 1994, January 1996, July 1997, and March 1998. The SRP reviewed the issues at its meetings in October 1997 and April 1998. A special meeting of the SRP was held on March 11, 1998, to hear testimony on health issues including the quantitative risk assessment from highly respected scientists invited by the Panel. Based on these reviews and information provided at scientific workshops and meetings, the SRP makes the following findings pursuant to Health and Safety Code section 39661:

Exposure related conclusions

1. Diesel exhaust is a complex mixture of gases and fine particles emitted by a diesel-fueled internal combustion engine.

2. The gaseous fraction is composed of typical combustion gases such as nitrogen, oxygen, carbon dioxide, and water vapor. However, as a result of incomplete combustion, the gaseous fraction also contains air pollutants such as carbon monoxide, sulfur oxides, nitrogen oxides, volatile organics, alkenes, aromatic hydrocarbons, and aldehydes, such as formaldehyde and 1,3-butadiene and low-molecular weight polycyclic aromatic hydrocarbons (PAH) and PAH-derivatives.

3 One of the main characteristics of diesel exhaust is the release of particles at a markedly greater rate than from gasoline-fueled vehicles, on an equivalent fuel energy basis. The particles are mainly aggregates of spherical carbon particles coated with inorganic and organic substances. The inorganic fraction primarily consists of small solid carbon (or elemental carbon) particles ranging from 0.01 to 0.08 microns in diameter. The organic fraction consists of soluble organic compounds such as aldehydes, alkanes and alkenes, and high-molecular weight PAH and PAH-derivatives, such as nitro-PAHs. Many of these PAHs and PAH-derivatives, especially nitro-PAHs, have been found to be potent mutagens and carcinogens. Nitro-PAH compounds can also be formed during transport through the atmosphere by reactions of adsorbed PAH with nitric acid and by gas-phase radical-initiated reactions in the presence of oxides of nitrogen.

4. Diesel exhaust includes over 40 substances that are listed by the United States Environmental Protection Agency (U.S. EPA) as hazardous air pollutants and by the ARB as toxic air contaminants. Fifteen of these substances are listed by the International Agency for Research on Cancer (IARC) as carcinogenic to humans, or as a probable or possible human carcinogen. Some of these substances are: acetaldehyde; antimony compounds; arsenic; benzene; beryllium compounds; bis(2-ethylhexyl)phthalate; dioxins and dibenzofurans; formaldehyde; inorganic lead; mercury compounds; nickel; POM (including PAHs); and styrene.

5. Almost all of the diesel particle mass is in the fine particle range of 10 microns or less in diameter (PM_{10}). Approximately 94 percent of the mass of these particles are less than 2.5 microns in diameter. Because of their small size, these particles can be inhaled and a portion will eventually become trapped within the small airways and alveolar regions of the lung.

6. The estimated population-weighted average outdoor diesel exhaust PM_{10} concentration in California for 1995 is 2.2 microgram per cubic meter ($\mu g/m^3$). Several independent studies have reported similar outdoor air diesel exhaust PM_{10} concentrations. The 1995 estimated average indoor exposure concentration is approximately 1.5 $\mu g/m^3$.

7. The population time-weighted average total air exposure to diesel exhaust particle concentrations across all environments (including outdoors) is estimated to be 1.5 $\mu g/m^3$ in 1995. This total exposure estimate may underestimate many Californians' actual total exposure because it excludes elevated exposures near roadways, railroad tracks, and inside vehicles. Near-source exposures to diesel exhaust may be as much as five times higher than the 1995 population time-weighted average total air exposure. It also excludes other routes of exposure to diesel exhaust, such as ingestion and dermal absorption.

8. Diesel engine exhaust contains small carbonaceous particles and a large number of chemicals that are adsorbed on to these particles or present as vapors. These particles have been the subject of many studies because of their adverse effects on human health and the environment. A recent study conducted for the Health Effects Institute showed that, despite a substantial reduction in the weight of the total particulate matter, the total number of particles from a 1991-model engine was 15 to 35 times greater than the number of particles from a 1988 engine when both engines were operated without emission control devices. This suggests that more fine particles, a potential health concern, could be formed as a result of new technologies. Further study is needed since the extent of these findings only measured exhaust from two engines and engine technologies.

9. The major sources of diesel exhaust in ambient outdoor air are estimated to emit approximately 27,000 tons per year in 1995. On-road mobile sources (heavy-duty trucks, buses, light-duty cars and trucks) contribute the majority of total diesel exhaust PM_{10} emissions in California. Other mobile sources (mobile equipment, ships, trains, and boats) and stationary sources contribute the remaining emissions.

10. Significant progress has been made as a result of federal and state regulations that have addressed particulate matter levels from diesel engines. Emissions of on-road mobile source diesel exhaust PM_{10} in California are expected to decline by approximately 85 percent from 1990 to 2010 as a result of mobile source regulations already adopted by the ARB.

11 The results of a study funded by the ARB at the University of California, Riverside, indicate that the diesel exhaust from the new fuel tested contained the same toxic air contaminants as the old fuel, although their concentrations and other components may differ. Further research would be helpful to quantify the amounts of specific compounds emitted from a variety of engine technologies, operating cycles, and fuel to characterize better any differences between old and new fuels and technologies.

Health effects associated with diesel exhaust

12. A number of adverse short-term health effects have been associated with exposures to diesel exhaust. Occupational exposures to diesel exhaust particles have been associated with significant cross-shift decreases in lung function. Increased cough, labored breathing, chest tightness, and wheezing have been associated with exposure to diesel exhaust in bus garage workers. A significant increase in airway resistance and increases in eye and nasal irritation were observed in human volunteers following one-hour chamber exposure to diesel exhaust. In acute or subchronic animal studies, exposure to diesel exhaust particles induced inflammatory airway changes, lung function changes, and increased the animals' susceptibility to infection.

13 A number of adverse long-term noncancer effects have been associated with exposure to diesel exhaust. Occupational studies have shown that there may be a greater incidence of cough, phlegm and chronic bronchitis among those exposed to diesel exhaust than among those not exposed. Reductions in pulmonary function have also been reported following occupational exposures in chronic studies. Reduced pulmonary function was noted in monkeys during long-term exposure. Histopathological changes in the lung of diesel-exposed test animals reflect inflammation of the lung tissue. These changes include dose-dependent proliferations of type II epithelial cells, marked infiltration of macrophages, plasma cells and fibroblasts into the alveolar septa, thickening of the alveolar walls, alveolar proteinosis, and focal fibrosis.

14. Studies have shown that diesel exhaust particles can induce immunological reactions and localized inflammatory responses in humans, as well as acting as an adjuvant for pollen allergy. Intranasal challenge with diesel exhaust particles in human volunteers resulted in increased nasal IgE antibody production and a significant increase in mRNA for pro-inflammatory cytokines. Co-exposure to diesel exhaust particles and ragweed pollen resulted in a nasal IgE response greater than that following pollen or diesel exhaust

particles alone. Effects of intratracheal, intranasal, and inhalation exposures of laboratory animals are supportive of the findings in humans. These effects include eosinophilic infiltration into bronchi and bronchioles, elevated IgE response, increased mucus secretion and respiratory resistance, and airway constriction.

15 Based on the animal studies, the U.S. EPA determined a chronic inhalation Reference Concentration value of 5 $\mu g/m^3$ for noncancer effects of diesel exhaust. This estimate takes into consideration persons who may be more sensitive than others to the effects of diesel exhaust. The report supports the recommendation of 5 $\mu g/m^3$ as the California Reference Exposure Level (REL) (Table 1). It should be noted that this REL may need to be lowered further as more data emerge on potential adverse noncancer effects from diesel exhaust.

16. Diesel exhaust contains genotoxic compounds in both the vapor phase and the particle phase. Diesel exhaust particles or extracts of diesel exhaust particles are mutagenic in bacteria and in mammalian cell systems, and can induce chromosomal aberrations, aneuploidy, and sister chromatid exchange in rodents and in human cells *in vitro*. Diesel exhaust particles induced unscheduled DNA synthesis *in vitro* in mammalian cells. DNA adducts have been isolated from calf thymus DNA *in vitro* following treatment with diesel exhaust particle extracts. DNA adducts have been shown to increase following inhalation exposure of rodents and monkeys to whole diesel exhaust. Elevated levels of DNA adducts have been associated with occupational exposure to diesel exhaust. Results of inhalation bioassays in the rat, and with lesser certainty in mice, have demonstrated the carcinogenicity of diesel exhaust in test animals, although the mechanisms by which diesel exhaust induces lung tumors in animals remains uncertain.

17. Over 30 human epidemiological studies have investigated the potential carcinogenicity of diesel exhaust. These studies, on average, found that long-term occupational exposures to diesel exhaust were associated with a 40 percent increase in the relative risk of lung cancer. The lung cancer findings are consistent and the association is unlikely to be due to chance. These epidemiolcgical studies strongly suggest a causal relationship between occupational diesel exhaust exposure and lung cancer.

18. Other agencies or scientific bodies have evaluated the health effects of diesel exhaust. The National Institute of Occupational Safety and Health first recommended in 1988 that whole diesel exhaust be regarded as a potential occupational carcinogen based upon animal and human evidence. The International Agency for Research on Cancer (IARC) concluded that diesel engine exhaust is probably carcinogenic to humans and classified diesel exhaust in Group 2A. Based upon the IARC findings, in 1990, the State of California under the Safe Drinking Water and Toxic Enforcement Act of 1986 (Proposition 65) identified diesel exhaust as a chemical "known to the State to cause cancer." The U.S. EPA has proposed a conclusion similar to IARC in their draft documents. The 1998 draft U.S. EPA document concluded similarly that there was sufficient animal evidence of carcinogenicity and that the human evidence was limited.

19. There are data from human epidemiological studies of occupationally exposed populations which are useful for quantitative risk assessment. The estimated range of lung cancer risk (upper 95% confidence interval) based on human epidemiological data is 1.3×10^{-4} to 2.4×10^{-3} $(\mu g/m^3)^{-1}$ (Table 2). After considering the results of the meta-analysis of human studies, as well as the detailed analysis of railroad workers, the SRP concludes that 3×10^{-4} $(\mu g/m^3)^{-1}$ is a reasonable estimate of unit risk expressed in terms of diesel particulate. Thus this unit risk value was derived from two separate approaches which yield similar results. A comparison of estimates of risk can be found in Table 3.

20. Based on available scientific information, a level of diesel exhaust exposure below which no carcinogenic effects are anticipated has not been identified.

21. Based on available scientific evidence, as well as the results of the risk assessment, we conclude that diesel exhaust be identified as a Toxic Air Contaminant.

22. As with other substances evaluated by this Panel and after reviewing the field of published peer reviewed research studies on diesel exhaust, additional research is appropriate to clarify further the health effects of diesel exhaust. This research may have significance for estimating the unit risk value.

23. The Panel, after careful review of the February 1998 draft SRP version of the ARB report, *Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant*, as well as the scientific procedures and methods used to support the data, the data itself, and the conclusions and assessments on which the Report is based, finds this report with the changes specified during our October 16, 1997, meeting and as a result of comments made at the March 11, 1998, meeting, is based upon sound scientific knowledge, methods, and practices and represents a complete and balanced assessment of our current scientific understanding.

For these reasons, we agree with the science presented in Part A by ARB and Part B by OEHHA in the report on diesel exhaust and the ARB staff recommendation to its Board that diesel exhaust be listed by the ARB as a Toxic Air Contaminant.

I certify that the above is a true and correct copy of the findings adopted by the Scientific Review Panel on April 22, 1998.

John R. Froines

John R. Froines, Ph.D
Acting Chairman,
Scientific Review Panel

TABLE 1

NONCANCER HEALTH VALUES APPROVED BY THE SCIENTIFIC REVIEW PANEL 1998

Compound		Health Value	Endpoint
Acetaldehyde			Respiratory System
Diesel Exhaust		5 $\mu g/m^3$	Respiratory System
Inorganic Lead		4.6×10^{-4} $(\mu g/m^3)^{-1}$	Cardiovascular Mortality
Perchloroethylene		35 $\mu g/m^3$	Alimentary System (Liver)

$\mu g/m^3$: microgram per cubic meter

TABLE 2

CANCER POTENCIES APPROVED BY THE SCIENTIFIC REVIEW PANEL FROM 1984 TO 1998
(in order of cancer potency)

Compound	Unit Risk $(\mu g/m^3)^{-1}$	Range $(\mu g/m^3)^{-1}$
Dioxins	3.8×10^{1}	2.4×10^{1} to 3.8×10^{1}
Chromium VI	1.5×10^{-1}	1.2×10^{-2} to 1.5×10^{-1}
Cadmium	4.2×10^{-3}	2.0×10^{-3} to 1.2×10^{-2}
Inorganic Arsenic	3.3×10^{-3}	6.3×10^{-4} to 1.3×10^{-2}
Benzo[a]pyrene	1.1×10^{-3}	1.1×10^{-3} to 3.3×10^{-3}
Diesel Exhaust	**3×10^{-4}**	**1.3×10^{-4} to 2.4×10^{-3}**
Nickel	2.6×10^{-4}	2.1×10^{-4} to 3.7×10^{-3}
1,3-Butadiene	1.7×10^{-4}	4.4×10^{-6} to 3.6×10^{-4}
Ethylene Oxide	8.8×10^{-5}	6.1×10^{-5} to 8.8×10^{-5}
Vinyl Chloride	7.8×10^{-5}	9.8×10^{-6} to 7.8×10^{-5}
Ethylene Dibromide	7.1×10^{-5}	1.3×10^{-5} to 7.1×10^{-5}
Carbon Tetrachloride	4.2×10^{-5}	1.0×10^{-5} to 4.2×10^{-5}
Benzene	2.9×10^{-5}	7.5×10^{-6} to 5.3×10^{-5}
Ethylene Dichloride	2.2×10^{-5}	1.3×10^{-5} to 2.2×10^{-5}
Inorganic Lead	1.2×10^{-5}	1.2×10^{-5} to 6.5×10^{-5}
Perchloroethylene	5.9×10^{-6}	3.0×10^{-7} to 1.1×10^{-5}
Formaldehyde	6.0×10^{-6}	2.5×10^{-7} to 3.3×10^{-5}
Chloroform	5.3×10^{-6}	6.0×10^{-7} to 2.0×10^{-5}
Acetaldehyde	2.7×10^{-6}	9.7×10^{-7} to 2.7×10^{-5}
Trichloroethylene	2.0×10^{-6}	8.0×10^{-7} to 1.0×10^{-5}
Methylene Chloride	1.0×10^{-6}	3.0×10^{-7} to 3.0×10^{-6}
Asbestos	1.9×10^{-4} (per 100fiber/m^3)	Lung: $11 - 110 \times 10^{-6}$ (per 100 fiber/m^3) Mesothelioma: $38 - 190 \times 10^{-6}$ (per 100 fiber/m^3)

$\mu g/m^3$: microgram per cub

TABLE 3

Comparison of Other Organizations' Estimated 95% Upper Confidence Limits of Lifetime Risk per $\mu g/m^3$ Diesel Particulate Matter from Risk Assessments Based on Epidemiologic Data with OEHHA Estimates

Method	Unit Risk/Range	Basis of Assessment	Reference
Epidemiologic analysis	3×10^{-4}	based on smoking-adjusted pooled RR	Smith, 1998
Epidemiologic analysis[b]	**3.6×10^{-4} to 2.4×10^{-3}**	**case-control study of Garshick et al., 1987**	**OEHHA, Part B, Section 7.3.3**
Epidemiologic analysis	**2.8×10^{-4} to 1.8×10^{-3}**	**cohort study of Garshick et al., 1988**	**OEHHA, Part B, Section 7.3.4**
Epidemiologic analysis	**1.3 to 7.2×10^{-4}**	**cohort study, time varying conc., roof (3,50) pattern**	**OEHHA, Part B, Appendix D**
Epidemiologic analysis	**3.8×10^{-4} to 1.9×10^{-3}**	**cohort study, time varying conc., ramp (1,50) pattern**	**OEHHA, Part B, Appendix D**
Epidemiologic analysis	1.4×10^{-3}	London transport study[c]	Harris, 1983
Epidemiologic analysis	2×10^{-3}	epidemiologic data of Garshick (top end of U.S. EPA's range)	U.S. EPA, 1998;
Epidemiologic analysis	1.3×10^{-4} to 1.3×10^{-2}	using smoking adjusted RR and exposures of 5 or 500 $\mu g/m^3$	OEHHA, Part B, Section 7.3; bracketed risk bounds

a) Bolded values are included in OEHHA's range of risk.
b) Obtained by applying Harris' slope of 5×10^{-4} $(\mu g/m^3 \times yr)^{-1}$ to California life table.

FF Principal Investigator/Program Director (*Last, first, middle*):

BIOGRAPHICAL SKETCH

NAME	POSITION TITLE
John R. Froines	**Professor**

EDUCATION (*Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training*)

Institution and Location	Degree	Year	Field of Study
University of California at Berkeley	B.S.	1963	Chemistry
Yale University	M.S.	1964	Physical-Organic Chemistry
Yale University	Ph.D.	1967	Physical-Organic Chemistry

RESEARCH AND PROFESSIONAL EXPERIENCE: Concluding with present position, list, in chronological order, previous employment, experience, and honors. Key personnel include the principal investigator and any other individuals who participate in the scientific development or execution of the project. Key personnel typically will include all individuals with doctoral or other professional degrees, but in some projects will include individuals at the masters or baccalaureate level provided they contribute in a substantive way to the scientific development or execution of the project. Include present membership on any Federal Government public advisory committee. List, in chronological order, the titles, all authors, and complete references to all publications during the past three years and to representative earlier publications pertinent to this application. DO NOT EXCEED TWO PAGES

Federal Public Advisory Committee:

1992 - 1995 Chair, Advisory Committee, Office of Technology Assessment
1995 - 1996 NIOSH, Search Committee for Director of Health Effects Laboratory Division
1995 - 1997 U.S. EPA, Common Sense Initiative, Electronics Sector
1997 - 1998 Federal Advisory Committee to DOE: Beryllium Standard
1999 - 2003 Member, National Toxicology Program Board of Scientific Counselors

Academic Interests:

Toxicology and exposure assessment. Research interests are in the qualitative and quantitative characterization of risk factors from environmental and occupational exposures. Special emphasis on exposure assessment and hazard surveillance research. Biomarkers/genetic toxicology in the study of chemical carcinogenesis and non-cancer toxicity. Studies on the carcinogenicity of arsenic, beryllium, and chromium. Health effects and toxicity of airborne particulate matter

Professional Experience:

1974 to 1977 Director, Division of Occupational and Radiological Health, Vermont Health Department, Barre, Vermont.
1977 to 1979 Director, Office of Toxic Substances Standards, Occupational Safety and Hlth. Administration, Washington D.C.
1979 to 1981 Deputy Director of National Institute for Occupational Safety and Health, Rockville, Maryland.
1981 to 1991 Associate Professor, Department of Environmental Health Sciences, UCLA School of Public Health.
1989 to Present Director, UCLA Center for Occupational and Environmental Health.
1991 to Present Professor, Department of Environmental Health Sciences, UCLA School of Public Health.
1994 to 1998 Chair, Department of Environmental Health Sciences, UCLA School of Public Health
1995 to Present Associate Director, NIEHS funded Southern California Environmental Health Sciences Center
1995 to 2000 Director, UCLA Pollution Prevention Education and Research Center (PPERC)
1995 to Present Principal Investigator, Fogarty Program for Occupational and Environmental Health
1995 to 2001 Member, Carcinogen Identification Committee, Cal/EPA
1997 to 1999 Acting Chairman, Scientific Review Panel, California Air Resources Board.
1999 to Present Chairman, Scientific Review Panel, California Air Resources Board.
1999 to Present Member, Scientific Advisory Board, Center for Vulnerable Populations Research
1999 to Present Director, Southern California Particle Center and Supersite
1999 to Present Member, Clean Fuels Advisory Group, South Coast Air Quality Management District
2002 to Present Member, Institute of Medicine, Roundtable on Environmental Health Sciences, Research, and Medicine
2002 to Present Member, Advanced Air Pollution Research Plan Steering Committee, SCAQMD
2003 to Present Chair, Asthma and Outdoor Air Quality Consortium Advisory Board, SCAQMD
2003 to Present Director, Centers for Environmental Quality and Health, UCLA School of Public Health

Awards:

American Industrial Hygiene Association – Southern California Section, *1999 Technical Achievement Award*
Coalition for Clean Air, *1999 Carl Moyer Award,* The 26th Annual Lester Breslow Distinguished Lecture, 2000.

The Center For Community Action and Environmental Justice, *Dr. Zweig Community Health Advocate Award*, 2002.

Publications:

Morgenstern H, Froines J, Ritz B, and Young B. Epidemiologic Study to Determine Possible Adverse Effect to Rocketdyne/Atomics International Workers from Exposure to Selected Chemicals. Report to the Public Health Institute, 1999.

Ritz B, Morgenstern H, Young BB, Froines J. Effects of Exposure to External Ionizing Radiation on Cancer Mortality in Nuclear Workers Monitored for Radiation at Rocketdyne/Atomics International. American Journal of Industrial Medicine, 35:21-31, 1999.

Froines JR, Barile R. Approaches to Addressing Occupational and Environmental Health Needs in Mexico. International Journal of Occupational Environmental Health, 5:208-212, 1999.

Ritz B, Morgenstern H, Froines J, Moncau J. Chemical Exposures of Rocket-Engine-Test-Stand Personnel and Cancer Mortality in a Cohort of Aerospace Workers. J Occup Environ Med, 41(10):903-910, 1999.

Okoji RS, Leininger J, Froines JR. Subchronic Toxicity Study of Sodium Arsenite in Methyl-Deficient Male C57BL/6 Mice. Arsenic Exposure and Health Effects, Proceedings of the 3rd International Conference on Arsenic Exposure and Health Effects, W. Chappell, C.O. Abernathy, and R.L. Calderon, editors, Elsevier Science, pg. 225-232, 1999.

Froines JR, Sabty R, Farrar C, Fernandez C, Meck N, Tan S, Ashley B. Occupational and Consumer Exposure to Hexavalent Chromium and Analysis of Occupational Exposure to Proposition 65 Chemicals. Final Report to the Public Health Trust, 1999.

Kim SY, Que Hee SS, Froines JR. Optimized Portable Cordless Vacuum Method for Sampling Dry, Hard Surfaces for Dusts. Applied Occupational and Environmental Hygiene, 15(6): 503-511, 2000.

Yu RC, Hsu K-H, Chen C-J, Froines JR. Arsenic Methylation Capacity and Skin Cancer. Cancer Epidemiology, Biomarkers and Prevention, 9:1259-1262, 2000.

The American Lung Association. Urban Air Pollution and Health Inequities: A Workshop Report, Proceedings of the Urban Air Pollution and Health Inequities Workshop, 1999. JR Froines - contributing member. Environmental Health Perspectives, 109 (Suppl 3):357-374, 2001.

Recio R, Robbins WA, Ocampo-Gomez G, Borja-Aburto V, Moran-Martinez J, Froines JR, Garcia Hernandez, RM, Cebrian ME. Organophosphorous Pesticide Exposure Increases the Frequency of Sperm Sex Null Aneuploidy. Environmental Health Perspectives, 109:1237-1240, 2001.

Kim S, Jaques PA, Chang M, Froines JR, Sioutas C. Versatile Aerosol Concentration Enrichment System (VACES) for Simultaneous In Vivo and In Vitro Evaluation of Toxic Effects of Ultrafine, Fine and Coarse Ambient Particles. Part I: Development and Laboratory Characterization. Journal of Aerosol Science, 32:1281-1297, 2001.

Froines JR, Cho AK, Hinds WC, Liu W-C, QueHee SS, Ritz B, Spear RC, Yu RC. Worker Exposure Assessment & Hazard & Medical Surveillance Programs. Final Report to NIOSH, 2001.

Yu RC, Hattis D, Landaw EM, and Froines JR. Toxicokinetic interaction of 2,5-hexanedione and methyl ethyl ketone. Archives of Toxicology, 75:643-652, 2002.

Sabty-Daily R, Luk KK, Froines JR. The Efficiency of Alkaline Extraction for the Recovery of Hexavalent Chromium (Cr^{VI}) From Paint Samples and the Stability of Cr^{VI} in a Paint. The Analyst 127 (6):852-858, 2002.

Okoji RS, Yu RC, Froines JR. Sodium Arsenite Administration via Drinking Water Increases Genome-wide and Ha-ras DNA Hypomethylation in Methyl-deficient C57BL/6J Mice. Carcinogenesis 23:777-785, 2002.

Li N, Kim S, Wang M, Froines J, Sioutas C, Nel A. Use of a Stratified Oxidative Stress Model to Study the Biological Effects of Ambient Concentrated and Diesel Exhaust Particulate Matter. Inhalation Toxicology 14:459-486, 2002.

Li N, Sioutas C, Cho A, Schmitz D, Misra C, Sempf J, Wang M, Oberley T, Froines J, Nel A. Ultrafine Particulate Pollutants induce Oxidative Stress and Mitochondrial Damage. Environmental Health Perspectives, 111 (4): 455-60, 2003.

Lippmann M, Frampton M, Schwartz J, Dockery D, Schlesinger R, Koutrakis P, Froines J, Nel A, Finkelstein J, Godleski J, Kaufman J, Koenig J, Larson T, Luchtel D, Liu LJS, Oberdorster G, Peters A, Sarnat J, Sioutas C, Suh H, Sullivan J, Utell M, Wichmann E, Zelikoff J. The EPA's Particulate Matter (PM) Health Effects Research Centers Program: A Mid-Course (2 ½ year) Report of Status, Progress, and Plans. Environmental Health Perspectives, 111(8): 1074-1092, 2003.

Cho AK, Di Stefano E, You Y, Rodriguez CE, Schmitz DA, Kumagai Y, Miguel AH, Eiguren-Fernandez A, Kobayashi T, Avol E, Froines JR. Determination of Four Quinones in Diesel Exhaust Particles, SRM 1649a and Atmospheric $PM_{2.5}$. Aerosol Science and Technology, in press 2003.

Yu RC, Teh HW, Jaques PA, Sioutas JR. Quality Control of Semi-Continuous Mobility Size-Fractionated Particle Number Concentration Data. In press, Atmospheric Environment. 2003